and Power Doppler Ultrasonography (PDUS). It is conceivable that local high-dose (40 mg of 6-methylprednisolone acetate) CS injection will have faster results than low oral CS treatment.

Their considerations about the real frequency of subacromial–subdeltoid (SA–SAD) bursitis and its role as diagnostic criteria for PMR was not an objective of our study and we think will be answered by an european league against rheumatism and ACR longitudinal multicentre study, the results of which will be presented soon. Differences in the selection of PMR patients and in the definition of US pathological inflammatory conditions, and the diverse probes and US machines utilized in the different studies may explain the wide range of reported prevalence in the literature of inflammation in articular and periarticular structures in PMR patients.

The persistence of US signs of inflammation even after 6 months of CS treatment is not that surprising and there are some explanations, as follows.

The sensitivity of US and PDUS in identifying signs of inflammation is significantly superior when compared with that of clinical examination. In rheumatological conditions, many studies have demonstrated that US and PDUS can identify inflammation in patients in clinical remission or in asymptomatic areas at enthesis or at joint level [4–7]. In our study we have demonstrated that also in PMR the increased sensitivity of US can identify patients with persistent US signs of disease activity but in clinical remission. Of the group of patients who entered our study, five still have signs of US shoulder periarticular inflammation after a mean follow-up of 26 months and three are CS free in persistent remission.

There is the possibility that some of our patients were incorrectly classified as PMR and that the persistence of signs of inflammation may have been related to the presence of a different inflammatory rheumatological condition. We know that a small percentage of patients diagnosed as PMR develop clinical findings of RA or other inflammatory articular conditions and that sometimes it is difficult to differentiate these syndromes at onset and only the patient follow-up will clarify the definitive diagnosis [8]. In the group of patients enrolled in our study, 12 had signs and symptoms of peripheral joint involvement at onset and 35% had US signs of gleno-humeral (GH) synovitis (mono or bilateral). At the time of the second examination after CS treatment, none had signs of peripheral arthritis and only one had persistent US signs of GH synovitis with Power Doppler signal. This patient was persistently RF negative and anti-cyclic citrullinated peptide negative, never developed peripheral erosions at follow-up and is now in complete clinical remission without treatment. Therefore, we are confident that PMR diagnosis in this particular patient was correct.

Disclosure statement: The authors have declared no conflicts of interest.

References


Comment on: Prevalence and incidence of rheumatoid arthritis in southern Sweden 2008 and their relation to prescribed biologics
Sir, Englund et al. [1] estimated the prevalence of rheumatoid arthritis using patient register from a large county in Sweden. The study provides important information on the epidemiology of RA. However, we were intrigued by
the discrepancy between the prevalence and incidence estimates. For example, in women the prevalence increases from 1.75% in those aged 60–69 years to ~2.1% in those aged 70–79 years. Assuming no major changes in the risk of RA over years, this increment in prevalence (0.35% over a 10-year period) suggests that the average annual incidence rate of RA in women aged 65–75 years is approximately 345 per 100,000 women. However, Englund et al. [1] calculated a much lower incidence rate of 130 per 100,000 women. This could be the result of too strict criteria for RA cases, such as requiring the diagnosis of seropositive arthritis or other RA on at least two separate occasions.

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Comment on: Prevalence and incidence of rheumatoid arthritis in southern Sweden 2008 and their relation to prescribed biologics: reply

Sir, We appreciate the comment by Chodick [1] recognizing the value of health care register data in the study of the epidemiology of rheumatic disease. However, we question his assumption and the calculation (based on the observed 0.35% increment in prevalence of RA between two adjacent 10-year-wide age strata), suggesting that the incidence of RA should be substantially higher than we reported [2].

We tried to replicate the author’s calculation, i.e. using an assumption of identical mean survival among RA patients and the general population (and no major changes in the risk of RA over the years), the expected incidence in this age category would actually be 10 times lower than that proposed in the comment, about 35 per 100,000 women per year.

Importantly, prevalence of chronic disease without true cure is a function of both incidence and survival, making inferring incidence from estimates of prevalence only hazardous. The incidence of RA in 65- to 79-year-old women that we reported in the article was derived directly from the observed number of incident RA cases during 2008 [2]. The estimate we obtained (135 per 100,000 women per year) is probably closer to the true incidence. An even higher increment in prevalence of RA than 0.35% between 60- to 69-year-old and 70- to 79-year-old women is likely suppressed by the increased mortality associated with the disease.

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